ABSTRACT

The present invention relates to novel modalities of treatment of diabetes, and other diseases caused by dysfunctional signal transduction by insulin receptor type tyrosine kinases (IR-PTK). Applicants discovered that IR-PTK activity may be modified by modulating the activity of a tyrosine phosphatase, and IR-PTK signal transduction may be triggered even in the absence of ligand. Methods for identifying compounds that, by modulating RPTPα or RPTPε activity, elicit or modulate insulin receptor signal transduction are also described.